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"Grass Disease" in Horses.*

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History. "Grass disease" of horses has only come into considerable prominence during the last thirteen or fourteen years. In accounts which have been given during the last quinquennium, it has usually been suggested that it was first seen at Barry, in Forfarshire, in 1907. This was in the practice of the late Mr. A. Spruell, of Dundee. From this centre it is usually described as having gradually spread until, now, cases have occurred in every county in the North East of Scotland. The South Eastern part of this area is still broadly the home of the disease, but odd cases are reported at intervals from remote parts of the country.

At this point it appears to be profitable to take stock of the position and search for evidence of the existence of the disease prior to the Barry Camp outbreak of 1907.

If proof can be advanced that the disease really did occur for the first time in this country on that occasion, a most important point will be established, as it will undoubtedly influence the course of investigations into its etiology.

Two very experienced practitioners with extensive knowledge of "grass disease" have told me that in looking back they can recall odd cases prior to 1907. There is no doubt, however, that it has taken an increased toll during the last ten or fifteen years and that for some years the losses have been of great economic importance.

Is there an Early History of Disease of this type in Veterinary Literature?

The literature concerning certain diseases which may or may not have much in common with "grass disease" is relatively large. They are usually described under three main titles, viz.:—

Epizootic cerebro-spinal meningitis.
Forage poisoning.
Borna disease.

There is no doubt that a whole range of diseases of this type, merging one into the other, can be described and it is difficult to say where one begins in the scale and the other ends. It is obvious that odd cases of disease, such as sporadic instances of stomach staggers, have sometimes been included in the descriptions of outbreaks. Also, it is certain that at extremes in the scale there are diseases which have no relationship to each other.

The claims which have been made regarding causal agents do not help enormously in the labour of sorting them out. Taking the whole class—if it be reasonable to speak of these diseases as a class—the most important causal agents incriminated have been moulds, *Bacillus botulinus*, streptococci and a filter-passing virus.

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As an illustration of the difficulty which competent observers have found in differentiating these diseases, it is interesting to quote the following remarks of Graham, Himmelberger and Pontius (1916)—"It seems quite difficult to make a differential diagnosis of cerebro-spinal meningitis (forage poisoning), Mohler; cerebro-spinal meningitis (encephalitis), also known by some as 'Kansas Horse Disease,' Udall; meningo-encephalitis (blind staggers), Haslam; encephalo-myelitis, Stange; epizootic cerebro-spinal meningitis, Hickman; and true forage poisoning, Pearson, as occurring in various parts of the United States; and of Borna disease, Hutyra and Marek, as occurring in Europe."

We will give a brief account of the diseases which fall within the above heads and then compare "grass disease" with them. In this way it will be easier to examine the relevant facts and attempt to answer the question under discussion.

Equine Epizootic Cerebro-Spinal Meningitis.

Wallis Hoare (1913) gives an account of this disease. It may be said at once that his description does not recall grass disease as we know it. He found on enquiry among practitioners of wide experience in the United Kingdom that such outbreaks had not come under their observation.

He cites various authors and discusses the conditions they had encountered and points out that there has been considerable diversity of opinion among investigators concerning the relationship of epizootic cerebro-spinal meningitis and Borna disease.

A good deal of what is said below concerning the history of "forage poisoning" and of "Borna disease" applies to cerebro-spinal meningitis. To avoid repetition we will proceed to the discussion of these diseases.

"Forage Poisoning."

Pearson (1901) investigated outbreaks and in one of them found that he could reproduce the disease by feeding experimental horses on a sample of the forage which had been fed to the sick animals.

He pointed out that the name usually applied to outbreaks of this type—cerebro-spinal meningitis—is wrong as there is no inflammation of the meninges, brain or cord.

He says: "Since this disease resembles the sausage poisoning and meat poisonings of man and carnivora, and since the observations, recorded above, show that the as yet undiscovered infectious or toxic principle resides in the food, I wish to suggest the name 'forage poisoning' as one that would be descriptive and accurate."

Mohler (1912) reviewed the question and gave a most interesting account of the history of the diseases which can be included within this title. His work has an added value in that it was written before it was known that *B. botulinus* can produce toxin in a purely vegetable pabulum and that therefore there was no direct suspicion that *B. botulinus* can be responsible for the disease.

A point common to most of these conditions is that over wide areas they have caused very heavy losses in the equine population. Perhaps the most important symptoms common to all of them are the interference with deglutition and afebrile course of the cases. To these, extreme prostration might usually be added. There is very rarely any indication in the literature of the percentage incidence in the exposed susceptible populations.

Mohler was careful to show that, in his opinion, certain outbreaks of disease which had occurred on the Continent of Europe and Great Britain and the "forage poisoning" of North America were the same disease. He says, "through the recent investigations of Grimm, Schmidt and others it has been quite definitely established that 'head disease,' 'Borna disease' and cerebro-spinal meningitis are one and the same, and Hutyra and Marek have accepted this. . . ."

This subject is considered further in the discussion on Borna disease.

In his description it is clear that the disease was not necessarily associated with the ingestion of stored food since he was as prepared to look for its origin in the pastures as in the stored fodder. He states that in some parts of the country the disease is known as "pasture disease." He found that it can be sporadic as well as enzootic. There was no conclusive evidence that the disease was communicated from one horse to the other. Attempts to transmit it by contact gave negative results.

"Forage poisoning" is said to be favoured by a long spell of dry weather ending in rain with high humidity. With the advent of cooler weather and lower humidity it stops.

Symptoms. (Mohler.) Usually disturbance of appetite, depression and weakness are the first manifestations observed. The disease then quickly declares itself. There is difficulty in swallowing, drooping of the head and sleepiness which may give way to excitement and attacks of vertigo. There are impairment of vision, loss of co-ordination, muscular twitching, cramp of certain groups of muscles, chiefly of the neck and flanks, and grinding of the teeth. Sometimes the animals try to push through obstacles. Some back into a corner and remain there. Others press the head against a wall or rest it on the manger. The temperature at the beginning is 103°—107°F The pulse is 40—90. Salivation occurs in some cases. Affected animals are usually lying down by the second or third day. The hind limbs may be paralysed. Coma or somnolence may be marked and death may occur in from eight hours to two to three weeks.

Other accounts of the symptoms agree fairly closely with those described above, with the exception that the disease is usually said to be afebrile. The great weakness is always emphasised. Graham, Himmelberger and Pontius (1916) frequently found impaction of the colon, but extensive intestinal stasis does not receive the attention that would be expected. Paralysis of the tongue is noted by some authors.

Lesions. Study of the lesions has not helped greatly in the elucidation of the etiology of the disease. They are frequently in marked contrast to the severity of the symptoms. There is usually a varying degree of inflammation in parts of the alimentary tract. The presence of stomach and intestinal parasites is frequently noted. The liver, spleen and kidneys are often congested and the bladder is often distended. Cystitis is sometimes present.

The condition of the central nervous system is variable. The brain is often normal, but the meninges are generally congested.

Buckley and Shippen (1917) say "Mohler (1914) attempted to explain the understanding that there is a toxic element in certain animal feeds that will produce the disease in contradistinction to the idea that poisonous plants are the cause."

Fischer (1922) does not accept the theory that botulism is the cause of all the forage poisoning occurring in the U.S.A.

"Borna Disease."

Mohler (1912) gives the following history of "Borna disease":—"About 100 years ago a fatal disease of horses appeared in Wurtemberg, called 'head disease,' owing to the pronounced manifestation of brain symptoms. It spread through certain sections of Europe in 1824—1828 and was called 'fever of the nerves.' In 1878 it was known in Saxony as 'nervous sickness' and within the next ten years assumed an epizootic character. It became so prevalent in and around Borna during the 'nineties' that it became known as 'Borna disease.' It had spread like a plague on two occasions in Belgium and had also exacted a heavy toll in Russia, Great Britain, Austria, Hungary and elsewhere. Its appearance in the U.S.A. is not a recent occurrence. It was reported in 1847 by Large and in 1869 by Liautard as appearing in both sporadic and enzootic form in several of the Eastern States. Since then, it has appeared periodically in many States in all sections of the country."

His remarks on the subject of the relation of "Borna disease" to "forage poisoning," etc., have already been quoted.

Wallis Hoare (1913) says:—"According to Friedberger and Fröhner, the disease known as Borna sickness in horses should be differentiated from cerebro-spinal meningitis as in the former affection there is an absence of lesions in the brain and spinal cord. Johne and Ostertag hold a similar opinion. Cadeac, however, states that the practitioners in the cantons of Borna, Merseberg, etc., have established the identity of the two affections. The clinical history of both appears to be identical, and as lesions of the nervous system may be absent in some cases of cerebro-spinal meningitis, or at least have not been discovered, and nothing definite has been ascertained as regards the bacteriology of either affection, it does not seem desirable to consider each disease separately."

Joest (1907) investigated "Borna disease" and found that although macroscopic lesions were absent, typical histological changes were present in the brain and spinal cord. He believed that the brain became infected by way of the lymph stream and not by the blood, and that infection travelled from the mucous membrane of the nasal cavity by the lymph vessels accompanying the olfactory nerves, these forming an unobstructed path to the pia mater of the olfactory lobes. He regarded the disease as "an acute, diffuse, infiltrating, non-purulent, meningo-encephalitis and myelitis of the lymphocyte type and chiefly mesodermal (vascular) in character." He described

special bodies in the form of nuclear inclusions in the olfactory lobe and large polymorphic ganglion cells of the hippocampus. These were not found in any other disease and when present were claimed to be diagnostic of "Borna disease," but "in individual cases they bear no relationship either as to size or number or as to localisation, to the inflammatory infiltrations."

Domizio (1919) discussed an outbreak among mules of the Italian Army in the Dolomite Alps. He described a sub-acute form of the disease in which there were marked spinal symptoms. He concluded that neither the diplostreptococcus of Johne (1896, 1897, 1900), Ostertag (1900), or Stohn (1910), nor any other organisms demonstrable by ordinary methods can be held to be the causal agent of the disease. His views were thus in agreement with those of Joest (1909—1913) and he believed that the disease was not a forage intoxication, but was in all probability set up by a filterable virus. He could not confirm the presence of Joest's intranuclear bodies. He stated that "Borna disease" was a distinct morbid entity and that it should not be confused with other kinds of meningitis or meningo-encephalitis.

Joest (1920) compared human encephalitis lethargica to "Borna disease." He said that the clinical symptoms and histological features of the brain correspond very closely in the two diseases.

Moussu and Marchand (1924) say that enzootic encephalitis or "Borna disease" does not appear to have been known in France prior to the war, but that it is not a rarity now. They consider that it was introduced by American horses.

They describe an outbreak they saw in which fifteen cases occurred in two months in a stud of eighty horses. It manifested itself

(1) as an encephalitis.
(2) as a myelitis.
(3) as a combination of these.

A brief Account of Bacteria, other than B. botulinus, which have been described in connection with Cerebro-Spinal Meningitis and Allied Conditions.

Ostertag in connection with "Borna disease" incriminated a streptococcus which he recovered from the blood, liver, urine, and brain of affected horses. Pure cultures inoculated subdurally or repeatedly injected into the bloodstream are said to have caused the disease.

Siedamgrotsky and Schlegel (1896) discovered cocci, frequently in the form of diplococci, in the cerebro-spinal fluid of cases of "Borna disease." On inoculation, fatal results were obtained in horses.

Johne (1896) isolated an organism from the cerebro-spinal fluid of cases of Borna disease, which he called *Diplococcus intracellularis equi*. Intradural inoculation induced symptoms of cerebro-spinal meningitis in horses.

Harrison isolated a streptococcus from the brain of a case of "forage poisoning" which was said to be identical with that found by Ostertag except that it formed capsules, would not grow on gelatine and was virulent for laboratory animals.

Christini (1909) reported finding *Micrococcus intracellularis meningitidis* of Weichselbaum (the cause of human cerebro-spinal meningitis) in the central nervous system in a sporadic case of meningitis in the horse and in the goat.

Each of the above authors considered his organism to be the cause of the disease he investigated and claimed that, on inoculation, it could reproduce it. Mohler (1912) says that some of the positive results are explained by the large quantity of turbid fluid injected under the dura mater (up to 5—10 c.c. of heavy emulsion).

Schmidt (1912) considered that the above-mentioned cocci are not the cause of the disease and that the etiology had not been discovered.

Grimm, working in Zwick's laboratory in Berlin, isolated streptococci not essentially distinct from those described by Ostertag. As cocci had been so frequently reported, he wondered if they are present in the brains of healthy horses and found that this may be so. In discussing this work, Mohler says that in the laboratory of the Bureau of Animal Industry, U.S.A., results were obtained that led to the belief that cocci are agonal invaders of the brain from the intestine.

Graham and Bruechner (1919), in referring to certain workers who have isolated cocci from the brain and spinal cord, express the same view. Kraus, Kantor and Quiroga in the Argentine isolated a diplostreptococcus from the brain in cases of "Borna disease" which they found capable of setting up a bacteræmia in horses and other animals. Neither the illness set up nor the lesions produced in the experimental cases were strictly comparable to the natural disease.

Murray (1924), working on human cerebro-spinal meningitis, says "a meningitis of horses, goats, and sheep is known which is caused by the *Diplococcus intracellularis equi* (Johne, 1896), of which the morphology and cultural characters differ very little from Weichselbaum's coccus, and a clinical, epidemiological and pathological account of the disease of Borna would amply describe cerebro-spinal fever of man (Cadeac, 1914)."

Graham and Himmelberger (1917), investigated an outbreak of "forage poisoning" in which they proved that oat hay which had been used by the diseased animals was uniformly poisonous when fed to horses. They isolated various types of microorganisms from the forage and among others a spore-forming, Gram-negative, ærobic bacillus which was pathogenic to horses and mules, less so for cattle, sheep and goats, while guinea pigs, rabbits and white mice were immune.

They say:—"The clinical manifestations observed in some experimental horses as the result of daily intravenous injections, rarely subsequently to feeding per os and rectum of this bacillus, and more constantly following the daily intravenous administration of sterile cultural filtrates in Uschinsky's medium, presented a striking resemblance to the symptoms evidenced by horses at the time of and subsequent to prostration as a result of eating oat hay."

Fungi as the cause of "Forage Poisoning," "Borna Disease" and Equine Cerebro-Spinal Meningitis.

Most of the earlier authors inclined to the view that toxic fungi were the cause of the disease. There were many attempts to set it up by feeding suspected fodder on which moulds or fungi had grown extensively, but with very few exceptions the results were negative.

Müller (1908) discussed fungi as the cause of an outbreak of disease affecting horses, cattle and sheep. Enquiries showed that in the year prior to the outbreak, the locality

had been severely attacked by fungi. Certain fields are said to have become absolutely black within a few days. The straw used as fodder for these animals had a mouldy odour and appearance and had been refused by the animals. One farmer mixed this straw with clean straw and his animals were severely affected. It was said that the fungus "represented a form of *Puccinia graminis*." Attempts to set up the disease with the fodder gave negative results.

Stange (1910) quotes Ferguson, who described cases due to smutted corn. Hickman studied cases in North Carolina, in which the cause was considered to be moulds on vegetation. McCarrol and McMullen describe an outbreak of "cryptogamic poisoning" in horses due to feeding mouldy beet tops.

Stange saw outbreaks he attributed to mouldy silage. It was toxic to horses in feeding experiments. He records: "At *post-mortem* no changes were present except a few petechiæ along the small intestine, a few infarcts in the kidney and slight softening of the brain." "Microscopical examination revealed the presence of mould in the mucosa and sub-mucosa of the small intestine, also mycelial threads growing between and around the renal tubules." He suspected that a mould was responsible for the death of these animals, and recorded the work which Buchanan carried out in connection with it. Buchanan had frequent occasion to examine the moulds common to improperly prepared silage. Numerous cultures were made and many moulds were isolated. Among them were several species of *Penicillium*, *Aspergillus*, *Mucor* and the mycelium of a Hymenomycete (probably a *Coprinus* and in one instance *Monascus*).

Mohler (1912) says that most investigators obtained negative results by feeding mouldy food, but some reproduced the disease in this way. Mayo (1890) fed a colt on mouldy corn and it developed the disease and died on the 26th day. Haslam (1910) said the Kansas outbreak of 1906 was produced by immature ears of corn infected by moulds, although the exact mould was not discovered. Horses fed on them developed typical fatal attacks of staggers in four out of seven cases. Klimmer comments on these negative results from feeding mouldy forage. Michener, Trumbower and Harbaugh attribute the disease to toxic fungi. Ceni, in Italy, states that moulds are capable of producing poisons, but only at certain stages of their growth. At other times they are inactive. He gives an instance in which mouldy baled hay was considered to have caused the disease. He says:—"Forage poisoning seems to be an autointoxication rather than an infection and due to certain chemical poisons or toxins formed by organismal activity. These toxins may be present when the forage is taken into the body or formed in the gastro-intestinal canal, and therefore the disease is a specific form of autointoxication. The nature of the substance is unknown."

Mohler isolated many fungi from damaged forage, and grew them in media in an endeavour to find a toxic agent. All gave negative results, but he points out that it is possible that laboratory conditions are not parallel to what occurs in the field.

Graham, Himmelberger and Pontius (1916) say that the corn in several outbreaks in 1911 was examined and found to be infected with fungi. An attempt to reproduce the disease with mouldy corn failed. Even so, mouldy food was considered to be closely identified with the disease.

Graham and Himmelberger (1917) quote Mohler (1914), Klimmer, Haslam (1910) and others, as suggesting the possibility that moulds may cause "forage poisoning." They say that the mould theory seems to be most plausible, but that experimental feeding and inoculations with certain moulds suggest that their relation to "forage poisoning" disease is insignificant. "The theory of toxin or poison producing organisms as a cause of this disease is supported by *post-mortem* findings in fatally afflicted animals in many forage poisoning outbreaks, but some of the moulds isolated from apparently poisonous forage, seem to possess limited toxic power . . ." "suggesting the . . . forage poisoning might be due to micro-organic life other than moulds or possibly to an agent even more remote."

Buckley and Shippen (1917) say that during an epizootic of forage poisoning studied, a mycologist investigated the vegetation upon which the horses grazed, particular attention being paid to the fungi, moulds, etc. A feeding experiment was carried out with samples of grasses and hay which were considered to be possibly contaminated with an infectious or toxic agent. The result was negative.

In one instance, on an 8,000 acre range in Montana, the mycologist found that there was intensive infection of certain grasses with *Claviceps purpurea*. A feeding experiment was tried with negative results, but was inconclusive, as, owing to a fall of snow, it was not possible to collect sufficient material.

Buckley and Shippen say that other investigators have reported forage poisoning in animals due to certain heavy growths of moulds on food and that these observations cannot be questioned.

Graham and Brueckner (1919) say that they were alive to the possibility that a toxic fungus might be the cause of the disease, but certain species resembling *Monascus purpureus*, *Fusarium* sp., *Aspergillus* sp. and *Diplodia zeae*, which they suspected, were found to be innocuous.

Botulism.

Bacillus botulinus was first isolated by Van Ermengen (1895) from a ham. Persons who had eaten the ham had contracted botulism. The disease was quite well known before that date, though the exact cause was not understood. For some years after the discovery, it was believed that the bacillus could only produce its toxin in a medium containing animal matter and that a purely vegetable medium would be unsuitable.

As the result of the work which Dickson carried out from 1915 onwards, it was found that a purely vegetable pabulum could supply all the conditions necessary for potent toxin production. This opened up a wide field of investigation and soon led to enquiry into the possibility that botulism might be the cause of equine "forage poisoning" epizootics.

Leuchs (1910) had found that two strains with which he was working were of different immunological types. Dickson and Burke confirmed this, the latter (1919) naming them types A and B respectively.

Buckley and Shippen (1917) working with a human strain showed that *B. botulinus* toxin could produce

symptoms of "forage poisoning" in horses, and Graham and Bruechner (1919) isolated *B. botulinus* from Indian corn silage, which had caused "forage poisoning" in horses. Toxin prepared from the bacillus produced an identical disease in mules.

Bengston (1922) isolated a culture of *B. botulinus* from the larvæ of *Lucilia caesar*. She proved that it was different from types of A and B already known, and designated it type C.

Seddon (1922) isolated a culture from a bone of a cow which had died of "Midland cattle disease" in Tasmania, six months previously. He proved that it was allied to *B. botulinus* and that its toxin produced the symptoms of botulism in experimental animals. He showed that it was different to types A and B and named it *B. parabotulinus*.

By 1919 Theiler and his colleagues, after working at "lamziekte" in cattle in South Africa for some years, had obtained, in impure culture, an organism reminiscent of but not identical to *B. botulinus*. It produced a toxin capable of causing the symptoms of lamziekte in experimental animals.

Finally (1926), they isolated the organism in pure culture, demonstrated that it is identical with Seddon's parabotulinus and named it *Clostridium parabotulinum bovis*.

Theiler and Robinson since then (1927) have isolated another culture of the organism from a rat found dead in the hay rack of the stall inhabited by two mules which contracted botulism. They showed that the tissues of the rat contained the toxin and that liquid excretions from it could have contaminated the mules' forage. They have shown that this culture differs from types A and B and from Seddon's parabotulinus and proposed that it be named *Clostridium parabotulinum equi*.

Weinberg (1926) in reviewing the literature on botulism points out that Graham and Bengston, presumably meaning Graham and Boughton (1924), showed that Bengston's type C and Seddon's *B. parabotulinus* are identical. He takes Theiler, Viljoen, Green, du Toit, Meier and Robinson (1926) and Theiler and Robinson (1927) to task over the names they have given to their strains, pointing out that they must be respectively *B. botulinus* types C and D.

The Action of B. botulinus Toxin. Dickson and Shevky (1923) pointed out that most investigators had followed Van Ermengen's lead in attributing the symptoms of botulism to lesions produced in the medulla oblongata and pons and especially in the nuclei of the various cranial nerves and of the anterior grey columns of the spinal cord. They had attempted to demonstrate organic changes in the cells of the nuclei of the cranial motor nerves which would afford anatomical cause for the characteristic disturbance of function.

They showed that the observations of the different investigators who studied the morbid histology of the tissues of individuals affected with botulism, varied considerably. Moreover, the changes which were described were not in accordance with the symptoms shown in the individuals examined. For example, although clinical observers agree that while there is marked general muscular weakness in botulism, there is rarely any true paralysis of the muscles of the extremities; yet most observers found that lesions were much more numerous in the nerve cells of the upper portion of the cord than in certain nuclei in the brain. That is, the parts served by the nervous system which was most severely injured were the least affected. They further pointed out that if the disturbances in function are dependent upon organic changes in the nerve cells, one cannot understand how function can be wholly re-established in the victims that do not succumb to the poisoning. They concluded that the method of histological study has not shown how botulinus toxin acts and that other methods must be employed.

They pointed out that acetyl choline appears to stimulate the portions of the nervous system, which are depressed in botulism. They tested the conduction of nerve impulses along the various nerves of the autonomic system in animals treated with *B. botulinus* toxin. Later they included studies on the function and rate of onset of fatigue of the skeletal muscles. Comparisons were made on normal animals.

These experiments showed that the action of *B. botulinus* toxin, types A and B, upon the nervous system is peripheral and not central. It affects the peripheral endings of the motor and secretory fibres of the parasympathetic system and exerts an influence on the motor fibres of the voluntary nervous system. The sympathetic nervous system is not affected.

The effect on the nerve fibres of the voluntary muscles appeared to be less severe as impulses were not completely blocked, as manifested by the rapid occurrence of fatigue. The sensory fibres of the peripheral nerves and the reflex arcs were not affected.

After Dickson and Shevky published their work, Cowdry and Nicholson (1924) carried out an investigation into the same question from a different angle. They made an independent careful study of the morbid histology of various tissues in animals experimentally dosed with botulinus toxin. They reviewed the literature of the subject in the following terms:—

The recent work of Dickson and Shevky and of Edmunds and Long (1923) had shown that the effects of botulism can be explained on the hypothesis that the real site of action of the toxin is upon nerve terminals and that the paralytic features may not be of central origin, as was formerly supposed.

This casts doubt on the previous histological work which had been carried out in connection with the disease.

Van Ermengen's discovery of *B. botulinus* and its toxin coincided with the work of some very brilliant neurocytologists (Golgi, Cajal, Nissl, etc.), and this led to intensive histological study of the central nervous system of botulism poisoned individuals. Considerable emphasis was laid on chromatolytic changes in Nissl's granules, and for some time the belief was held that the other manifestations of the disease were secondary phenomena. There was, however, no general agreement as to the distribution of the lesions. Some considered them to be most marked in the spinal cord and to decrease in severity as the higher centres are reached, while others emphasised particularly the changes in the nuclei of origin of the cranial nerves, not even looking for lesions elsewhere.

Cowdry and Nickolson say that none of the papers they have read is provided with good enough illustrations for

them to ascertain with any degree of accuracy the real nature of the alterations observed. A further difficulty in estimating the value of the earlier work is that there are no available records supplying quantitative data of the percentage of nerve cells altered and the exact degree of change so that the personal equation of interpretation is a large one in each instance. On this account they consider that some of the alleged modifications may fall within the normal range of variation.

There is more agreement regarding the changes in the vascular system. The majority of investigators have reported marked congestion of the blood vessels with small hæmorrhages into the brain substance, and Dickson has concluded that a type of thrombosis is pathognomonic of botulism.

Cowdry and Nicholson found that there were no changes in the Nissl's granules or in the nuclei of the nerve cells which were not likewise observed in apparently normal controls. "Except for a slight degree of vascular engorgement, all the lesions found in the brains of the animals examined were readily susceptible of some explanation other than that they are produced by the direct action of the toxin upon the central nervous system. This absence of microscopic evidence of a central action of the toxin is not of course conclusive proof that the cells in the brain are not primarily involved; because it is quite conceivable that, owing to the delicacy of the nervous mechanism, the toxin might well act centrally without leaving any traces microscopically visible. But the observations do tend to show that, upon the histological side, there is no evidence inconsistent with the results of physiological experiments indicating that the site of action of the toxin is upon peripheral nerve terminals."

A Filter-Passing Virus.

Moussu (1924) and his collaborators investigated enzootic equine encephalitis in France. They claim that by inoculating suspensions of brain matter from infected horses into the anterior chamber of the eye of the rabbit, a transmissible disease can be maintained in series and that the agent is a filter passing virus. They claim further that they reproduced the disease in horses by intra-ocular inoculation of brain matter from a rabbit in the series.

Zwick and Seifried (1925) found that the disease is transmissible from the horse to the rabbit by intracerebral inoculation of cerebral substance. They found that it cannot be transmitted to other animals by any other mode of inoculation. When transmitted from the horse to the rabbit, the period of incubation lasts on the average about four weeks. In later passages from rabbit to rabbit it is three weeks. When first passaged in this way the illness lasts thirteen days and later varies from ten to eighteen days.

The symptoms of the transmitted disease are distinctly analogous to those of "Borna disease" in the horse.

The work of Zwick and Seifried (1926), Beck and Frohbose (1926) and of Ernst and Hahn (1927) confirms the existence of a filter passing virus.

All this work is most interesting. It is based on the work carried out by Levaditi and his co-workers (1921 onwards) on poliomyelitis, encephalitis lethargica and other conditions which they showed were caused by filter passing viruses having the common property that they possess a special affinity for tissues derived embryologically from the ectoderm.

"Grass Disease."

Occurrence.

Season. "Grass disease" is primarily a seasonal condition. Odd cases do occur at any time throughout the year, but the actual season is very narrowly defined. June is emphatically the worst month, and it is probably safe to say that 60 per cent. of the cases occur in that month. A further very important feature in the incidence of the disease is that the majority of the cases that occur in June are of the peracute or acute type and that the occurrence of cases of these two types is almost limited to June. It is to be borne in mind that over the greater part of the seriously affected country, the horses are turned out at night within a few days of the term day, the 28th May. It is within ten days to a fortnight of the time when most of the horses are put out at night that cases begin to be numerous.

The disease runs on during July, but most of the cases that occur during this month are of the subacute or chronic type. A few cases occur in August.

Weather. It is often said that the weather has an influence on the disease. Fine, dry, warm weather with frosty nights is said to be a predisposing factor, while it is said to stop after rain.

The Occurrence of "Grass Disease" in Stall-Fed Horses. It is to be understood that "grass disease" is essentially a disease of horses at pasture or fed on cut grass. At least one very experienced practitioner, with an extensive knowledge of the disease, asserts that whenever a case has occurred in a stall-fed horse, he has been able to ascertain that it could possibly have eaten green grass. He has been able to obtain a history such as that it had delivered a load at a farm and was left standing for some time.

Instances are known in which horses kept in a stable and given a diet free from green grass have contracted the disease; but these are rare.

Symptoms. These vary considerably with the type of case and the stage of the disease. The symptoms of fundamental importance are, lack of pyrexia in most cases, difficulty in swallowing, intestinal stasis, tendency to accelerated heart beat, icteric appearance of the conjunctiva, tremors of the muscles behind the shoulder and over the stifle joint, intermittent patchy sweating, snoring respiration in some types and nasal discharge. Regurgitation of ingesta from the stomach commonly occurs.

The patient is usually rather somnolent, but is easily roused. Intelligence is unimpaired. In geldings the penis is usually pendulous. There may be retention of the urine. Salivation occurs intermittently in some acute cases, but not in others. The pupils are usually unaffected.

In cases which live for more than a few days after the onset of the symptoms, there is an exceedingly rapid loss of condition. The abdomen is tucked up to an exaggerated degree. Diarrhœa is a feature of a small minority of cases. There is no paralysis of the tongue, general tendency to inco-ordination of movement or to mania or early loss of consciousness.

LESIONS. These naturally vary with the type of case.

Acute Type. Tympany is frequent. The stomach is usually distended with fluid, and the mucous lining is intensely congested. Rupture is an occasional sequel to the distension and attempts to vomit. There are usually liquid and gas in the small intestine and more or less congestion in places. The colon is very dilated and filled with a dry hard mass of impacted ingesta. In the cases in which there is complete impaction, the mass is always described as being so hard that it can scarcely be indented by the fingers. The mucous membrane is congested in places and the impacted food mass is covered by a thin jet-black layer. The fæcal matter is very adherent to the bowel wall. There is some degree of congestion of the liver, spleen and kidneys, but the spleen is of normal size.

There is great variation in the appearance of the central nervous system. In many cases the tissues appear to be normal. In others, there is congestion of the meninges. In some cases there are pin-point hæmorrhages throughout the substance of the brain. The cerebro-spinal fluid is always of normal appearance.

Subacute Type. There is emaciation. Impaction is absent, and the volume of ingesta in the colon is abnormally small and of pultaceous consistency. The spleen is abnormally large and of leathery consistence.

There is often an intensely inflamed area in the naso-pharynx, and food material may be found in the posterior nares. As a consequence, necrosis of the mucous membranes and even of the ethmoid bone is not uncommon.

The brain and spinal cord show the same variations as the acute cases.

INCIDENCE.

Percentage incidence of the disease in the equine population in the "risk" area.

It is important that this point be established. Most of the horses in the "risk" area are insured. The premium for all risks including "grass disease" was originally four and a half per cent. Two-thirds of the value was paid for losses from "grass disease" and full value for losses from other causes. Later it was put up to six and a quarter per cent., full value being payable on "grass disease" losses. For the last two years it has been lowered to five per cent. This premium does not, of course, give a fair estimate of the incidence of the disease as it is a flat rate over the whole country and presumably the horses in the "risk" area only represent a fraction of those insured. Nairn (1923) stated that the grass disease mortality for the County of Forfarshire was about two per cent.

Age and Sex. There is no evidence that age or sex has any effect on the incidence of the disease.

Immunity. There is no evidence that an attack of the disease confers immunity.

HAS GRASS DISEASE ANY RELATIONSHIP TO THE DISEASES ENUMERATED ?

A description of any of the diseases discussed fails to recall "grass disease" as we know it. Although to a certain extent they are seasonal, a review of the literature fails to leave an impression that there is such regularity in this respect as there is in the incidence of grass disease. Excitement or even mania is a feature of "Borna disease," and is frequently said to occur in cases of American "forage poisoning." While excitement is admitted to occur in some cases of grass disease, it is never a mania and in any case this train of symptoms only occurs in a minority of affected animals. Inco-ordination of movement and loss of consciousness are features of both forage poisoning and Borna disease, but are not symptoms of grass disease.

In forage poisoning and some cases of experimental botulism, paralysis of the tongue is a striking symptom. It never occurs in grass disease.

The most striking feature common to all of this class of diseases is a degree of bulbar paralysis, but there is considerable variation in the degree to which the bulb is affected. At the same time there is evidence that other parts of the central nervous system are deranged in some of the conditions.

It is established that the toxin of the different types of *B. botulinus* can cause a train of symptoms attributable to a partial bulbar paralysis. It is similarly established that in the case of one of these conditions, "Borna disease," a filter passing virus is present.

There is strong evidence that in all these conditions, types of cocci are present in the brain, cerebro-spinal fluid or both, and that they complicate the disease processes.

It appears to be not unreasonable to suggest that further work may show that these diseases may be classified as follows :—

1. An encephalitis or encephalo-myelitis caused by a filter passing virus.
2. *B. botulinus* intoxication.
3. Pure cerebro-spinal meningitis.

In addition, "grass disease" and many outbreaks of "forage poisoning" would fall within still other categories.

The available evidence indicates that "grass disease" is a clinical entity.

POSSIBLE CAUSES OF GRASS DISEASE WHICH REQUIRE FURTHER INVESTIGATION.

A Plant Poison. Considerable search has been made with negative results on "grass disease" pastures for any evidence of poisonous plants. At one time it was suspected that the alsike clover in the pastures was poisonous, but no evidence could be produced to support this theory.

If a poisonous plant were concerned, it would in most cases be equally poisonous when dried with hay, and in that event the disease would not be uncommon in stall fed animals and during the winter. It is not, of course, impossible for a poison to be concerned which is only dangerous during a short season and is destroyed on drying.

It may be said that with our present knowledge a plant poison might in some circumstances offer the easiest explanation of the curiously haphazard and low incidence of the disease in the susceptible population.

The actual method of spread has not yet been sufficiently closely followed. It has been noted in some cases that it has spread along thoroughfares and in others along the courses of rivers. A point which is very difficult to understand in connection with all the existing possible hypotheses is the very localised distribution of the disease.

If a poisonous plant were concerned, it would be expected to have opportunities for dissemination to such an extent that it would have a wide distribution by now, or, if it only

existed with difficulty, it might be expected to have died out.

A Filter-Passing Virus. The known viruses are all spread by direct contagion or by the agency of an insect vector. When a micro-organism has to exist through the agency of a vertebrate and invertebrate host, it usually has to infect a relatively high percentage of both susceptible populations, or it is in constant danger of dying out.

There does not appear to be much evidence that grass disease is spread by direct contact. Usually the incidence is opposed to evidence of direct infection. If a close enquiry be made, however, there are often small traces of evidence in favour of such a view. For example, it is often noticed that, if two horses are attacked on the same farm, they are a pair. Even when at pasture the pairs of horses tend to keep together. Other things being equal, when one of a pair becomes infected, if there is any direct infection, the fellow would be expected to have had most opportunity of contracting it.

This particular piece of evidence could be claimed to point to common infection from the same source, the low incidence being explained by scanty presence of the causal factor in the neighbourhood concerned. As instances of this, a very scanty poisonous plant or very limited and scanty botulinus contaminated areas of ground would afford an adequate explanation.

Similarly, it is often said that when the disease occurs in a new area, a neighbouring farm is likely to have a case even though only one or two horses are attacked on each farm.

If it be held that a filter passing virus carried by an insect vector is the agency responsible for the disease, it will almost be necessary to postulate a hypothetical "reservoir" of the virus in the locality and to assume that a relatively high percentage of the horses in the "risk" area contract the disease without showing visible symptoms.

Botulism. It is hard to see where *B. botulinus* has an opportunity to multiply on pastures. It can, of course, live in symbiosis with ærobic micro-organisms if a suitable pabulum be present. It is barely conceivable, but exceedingly unlikely, that rotting vegetation such as the frosted leaves of rye grass could in certain circumstances supply a small nidus here and there with sufficient nutriment.

Theiler and Robinson's recent discovery of course opens up much a wider field as the bodies of small animals might occur in all sorts of pasture and develop a potent toxin in the course of decomposition.

The symptoms of "grass disease" do not closely coincide with the symptoms recorded as those of botulism in equines.

A Streptococcus. Most of the investigators who have worked on this type of disease have encountered streptococci in the central nervous system. These micro-organisms are sufficiently constantly present to intrude very prominently on the notice of the pathologist, as, besides growing in suitable culture media, they frequently cause the death of experimental animals inoculated with material from the central nervous system of cases of the disease.

The evidence suggests that these micro-organisms are of secondary importance in the disease. They appear to be incapable of setting up a natural attack under experimental conditions.

A Fungus. There is no valid reason why a fungus should not be responsible for grass diease. A considerable amount of work has been carried out in the search for such an agent as the cause of "forage poisoning," but all the efforts have been unsuccessful.

Any other Micro-organisms. There does not at present appear to be any evidence to suggest that any other type of micro-organism plays a primary rôle in this connection.

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(Continued at foot of first column next page.)

Acute "Grass Disease": An Interpretation of the Clinical Symptoms.*

By J. RUSSELL GREIG, Professor of Medicine, Royal (Dick) Veterinary College, Edinburgh.

In investigating the cause of an obscure epizoötic it may be profitable to study the effects which the disease produces upon the functions of the organs as well as upon their structure.

The evidence furnished to the pathologist on a *post-mortem* examination of the cadaver may be important enough, but valuable clues to the nature of the disease may emerge from a close study of these signs of disordered function which we call clinical symptoms. Although the pathologist encounters the conditions which have caused death, it does not always follow that such conditions can be accepted as manifestations of the *nature* of the disease. In other words, those signs which result from the early, as distinct from the ultimate, effects of disease may, in their proper interpretation, furnish us with important information as to the origin of the pathogenesis.

In acute "grass disease" we are furnished with a clinical picture composed of definite symptoms, and, although in individual cases this or that symptom may be represented in varying degrees of intensity, the picture is essentially the same in the details of its composition.

For reasons which will appear later, there seems good cause for believing that the symptoms in the acute type represent the effects of functional nervous derangement, and, before proceeding further, it would be well to glance at the structure and function of the central nervous system.

The nerve fibres which compose the conducting mechanism of the central nervous system fall into two main groups :—

(1) *Afferent fibres* which conduct messages from all parts of the body to the central nervous system.

(*Continued from previous page.*)

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(2) *Efferent fibres* which conduct messages from the brain and cord to the peripheral parts of the body.

Efferent nerve fibres may be primarily classified as :—

(*a*) those which innervate voluntary muscle and are therefore under the governance of the will, and concerned in voluntary movement.

(*b*) those which govern the processes of the body over which there is no voluntary control.

The vital processes, such as cardiac contraction, gland secretion, intestinal movement, etc. are not under the control of the will and represent a system of self government or autonomy, and the nervous mechanisms which control such functions form what is known as the autonomic nervous system.

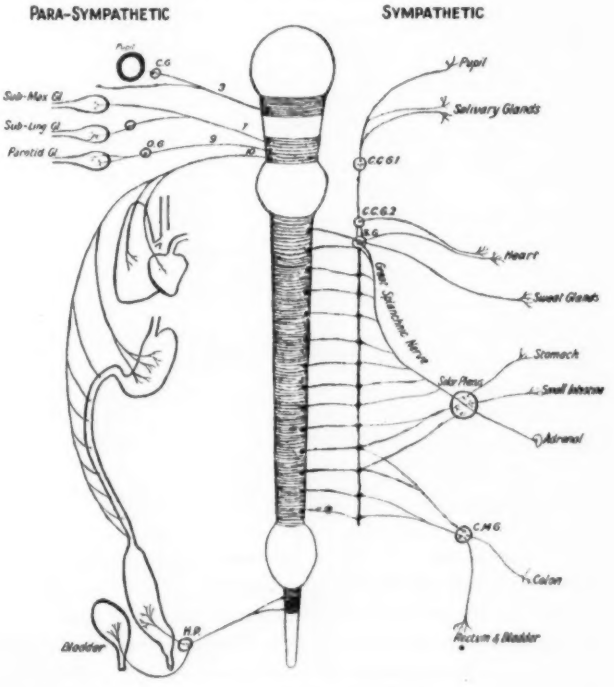
The autonomic nervous system includes two distinct mechanisms :—

(1) The PARASYMPATHETIC, composed of

(*a*) fibres issuing from the brain stem in the 3rd, 7th and 9th cranial nerves, together with all the efferent fibres in the 10th cranial nerve (cranial autonomic).

(*b*) fibres issuing from the anterior roots of certain sacral nerves (sacral autonomic).

(2) The SYMPATHETIC, which originates in fibres given off from the cord in the anterior roots of the spinal nerves between the cervical and sacral swellings.



PARA-SYMPATHETIC.

C.G. Ciliary Ganglion.
O.G. Otic Ganglion.
H.P. Hypogastric Plexus.
3, 7, 9, 10. The figures refer to the Cranial Nerves.

SYMPATHETIC.

C.C.G.1. Cranial Cervical Ganglion.
C.C.G.2. Caudal Cervical Ganglion.
S.G. Stellate Ganglion.
C.M.G. Caudal-mesenteric Ganglion.

Scheme of the general arrangement of the Autonomic Nervous System, the distribution of Sympathetic and Parasympathetic portions being contrasted on the two sides of the diagram. (Partly after Langdon Brown).

These two systems, the parasympathetic and the sympathetic, are mutually antagonistic since, in the main,

the effects produced by their respective stimulation are opposed; thus, while the parasympathetic is motor to the intestine, the sympathetic inhibits its peristaltic movement; the parasympathetic inhibits the heart's action, the sympathetic accelerates it. It is by this process of counteraction that a condition of "balance" in the vital functions is largely maintained.

The effects produced by the respective stimulation of the parasympathetic and sympathetic systems may be briefly shown in the following table:—

TABLE No. 1.

AUTONOMIC NERVOUS SYSTEM.

PARASYMPATHETIC.		SYMPATHETIC.
Contract	Pupil	Dilate
Inhibit	Heart	Accelerate
Secrete (thin and watery)	Salivary Glands	Secrete (thick and glairy)
Motor	Stomach	Inhibit
Nil	Pyloric Sphincter	Constrict
Motor	Intestines	Inhibit
Nil	Ileo-Cæcal Sphincter	Constrict
Contract	Bladder	Relax
Nil	Sweat Glands	Secrete
———	Superficial Muscles	Tremor
Nil	Voluntary Muscles Generally	Increased Plastic Tonicity
Constrict	Bronchi	Relax
Nil	Anal Sphincter	Constrict

The condition of balance which is maintained by the counteraction of these two systems may be upset by various factors which elicit a specific effect of stimulation or depression on one or other of the divisions of the autonomic nervous system. These factors include such varied substances as vegetable alkaloids, toxins and endocrine secretions. Thus:—

Arecoline, Physostigmine, Pilocarpine, Acetyl-choline } Stimulate parasympathetic.

Atropine, Hyoscyamine } Depress parasympathetic.

Adrenaline, Tyramine } Stimulate sympathetic.

Ergotamine — Stimulates, but later depresses sympathetic (motor fibres).

Certain agents, then, are known to excite one or other of the systems, and in so doing they produce a condition of *relative* depression in the opposite system. Conversely, those agents which depress one system produce a condition of *relative* activity in the other.

A condition of dominance in the parasympathetic is spoken of as VAGOTONIA, while SYMPATHICOTONIA is used with reference to excessive activity of the sympathetic.

"Grass disease" occurs in acute, sub-acute and chronic forms, and, in the present state of our knowledge, all three forms are generally regarded as representing differing degrees in the manifestation of one essential pathological condition.

In a study of the symptomatology, such as one is here attempting, the acute form alone will be considered, because in the more chronic types secondary symptoms develop which make for confusion and so obscure the issue.

As has already been indicated, acute "grass disease" is remarkable in that it is always presented clinically as a definite and complete syndrome; that is to say, its recognised clinical symptoms are practically always present (although in variable degree), in all cases.

In the acute form there is little or no fever; the pulse is accelerated; a variable amount of thick glairy saliva is secreted; there is difficulty in swallowing, and regurgitation of ingesta may occur; the intestines are in a condition of stasis which approaches the absolute; there is frequently retention of urine; sweating and the development of fine muscular tremors are also observed; there is increased muscular tonus which may amount to tonic contraction, especially in the lumbar and gluteal groups (this is evidenced by the impression of hardness or "resistance" obtained by digital palpation); the sphincters of the alimentary tract are in tonic spasm.

This last statement may require some explanation. The spasm of the *pyloric sphincter* is evidenced by the acute distension of the stomach with fluid. Further, it has been observed by Nairn (private communication), that large doses of strychnine, if administered by mouth, produce no toxic effects in acute "grass disease"; the strychnine being presumably confined to the stomach and its absorption by the small intestine thus prevented.

Spasm of the *ileo-cæcal sphincter* is shown by the sudden and sharp change in the nature of the intestinal contents at the ileo-cæcal junction; that is, the approximate normality of the small intestine as compared with the almost completely dry condition of the cæcum and large bowel.

The spastic condition of the *anal sphincter* is obvious on rectal examination of the patient.

On *post-mortem* examination no definite structural change has been described as constant. The distension of the stomach with fluid and the sharp change which occurs in the gut at the ileo-cæcal junction have already been mentioned. The symptom of regurgitation may fairly be regarded as a secondary effect dependant upon the gastric distension.

It will be seen that the symptoms of acute "grass disease" appear to be wholly referable to functional disorder, in that they develop without appreciable cause, and cannot be associated with any recognisable organic lesion.

Now, let us compare the effects produced by sympathetic stimulation with the symptoms of acute " grass disease."

TABLE No. 2.

Effects of Sympathetic Stimulation.		Symptoms of Acute Grass Disease.
Dilated	Pupil	Normal
Accelerated	Heart	Accelerated
Salivation ; (thick and glairy)	Salivary Glands	Salivation ; (thick and glairy)
Inhibition	Stomach	Stasis
Constriction	Pyloric Sphincter	Constriction
Inhibition	Intestines	Stasis
Constriction	Ileo-Cæcal Sphincter	Constriction
Relaxation	Bladder	Frequently retention of urine
Secretion	Sweat Glands	Sweating
Tremor	Superficial Muscle	Tremor
Increased plastic tonicity	Voluntary muscle	" Resistance " to palpation
Relaxation	Bronchi	*Unknown*
Constriction	Anal Sphincter	Constriction

It will be observed that a comparison of the symptoms of acute " grass disease " with those of sympathicotonia presents a remarkable similarity ; indeed the similarity is so close as to appear beyond the possibility of chance. The one exception would seem to be provided by the pupil. Some observers have stated that the pupil was dilated in acute " grass disease," but careful examination of a number of cases would indicate that the pupil is approximately normal.

It has been generally believed that sympathetic stimulation, induced by the intravenous injection of adrenaline, caused appreciable dilatation of the pupil in the horse. This does not agree with the few observations I have made on the point in the course of practice, and, through the kindness of Professor Pool and his assistant Mr. Brownlee, facilities were recently given me to put the matter to the test of experiment.

The pupils of three horses were carefully measured, and thereafter a solution of adrenaline chloride, 1 in 10,000 in doses of 40, 50 and 60 c.c. was administered intravenously to each horse respectively. In each case an intense sympathicotonia was developed, but in no case was there any appreciable change in the pupil. By way of comparison, after an interval of forty-eight hours one of the test horses was given ½ gr. atropine sulphate intravenously ; a pronounced mydriasis developed within a few minutes, and the pupil remained dilated for several hours.

Professor A. J. Clark, of Edinburgh, and Professor Pool, obtained similar results in two cases, in that they observed that the sympathicotonia consequent upon intravenous injection of adrenaline, although evidenced by pronounced cardiac acceleration, rise in blood pressure, sweating, tremor, etc., elicited no perceptible effect upon the pupil.

It would seem, then, that (in contradiction to established belief) the pupil of the horse does not appreciably respond by dilatation to the sympathetic stimulation induced by adrenaline. On the other hand, dilatation is readily induced by parasympathetic depression.

The normality of the pupil in " grass disease " may therefore be regarded as the exception which goes to prove the rule.

While my interpretation of acute " grass disease " as a sympathicotonia may be admitted, it may also be argued that the condition is a relative, and not an absolute, one, *i.e.*, that it arises from a depression of the parasympathetics rather than from a stimulation of the sympathetics.

On reference to Table No. 1, it will be seen that certain structures are innervated by the sympathetic only, *e.g.*, the intestinal sphincters and the sweat glands. Now, if an *absolute* depression of the parasympathetics occurred in " grass disease," these structures would not be activated. The experiments referred to above in which the pupil dilated to parasympathetic depression and not to sympathetic stimulation would appear to lend further support to this view.

In this connection it seems to me that the height of blood pressure and the degree of sugar tolerance which obtain in " grass disease " should be determined.

If, then, as appears to be the case, the symptoms can be interpreted as representing an excessive stimulation of the sympathetic system, one is led to enquire what type of factor is capable of producing such stimulation.

We know that the normal stimulation of the sympathetic in health is largely maintained by the adrenal medulla, and with this endocrine gland the thyroid is believed to co-operate. It is probable that the symptoms arise from overactivation of this group ; but this overactivation must, in turn, be dependant upon some primary factor. That such factor is of exogenous origin is clearly shown by the well known geographical distribution of the disease.

(*a*) *Poisonous Plants* : At least one plant, *Ephedra vulgaris*, is known to produce a substance, ephedrine, which elicits powerful sympathetic stimulation. The plant is not, however, a native of this country. The possible connection between the disease and plant poisoning has been, hitherto, necessarily conducted in a somewhat desultory manner. A thorough systematic enquiry seems highly desirable.

(*b*) *Fungi* : Ergot contains certain active substances, such as ergotoxin and tyramine, which are sympathetic stimulants. It is not suggested that ergotism is necessarily related to the disease, but it is possible that other fungal parasites of grass may contain toxins of a similar nature.

(*c*) *Bacterial Toxins* : So far as one is aware no bacterial toxin is known to be a specific sympathetic stimulant,

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The Veterinary Record.

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"GRASS DISEASE" IN HORSES.

The proceedings of the 1927 Annual Meeting of the Scottish Branch of the N.V.M.A. will be read with interest. This is the third occasion upon which the subject of "Grass Disease in Horses" has been discussed by this Branch, the first being in 1921, when Professor Gaiger expressed his belief that "grass disease" and Borna disease were identical; the second in 1922, when Dr. Tocher and his collaborators announced their opinion, which first took shape in 1919, that "grass disease" is equine botulism.

Since the comparison between "grass disease" and Borna disease was made, scientific investigation into Borna disease has been considerable, and our knowledge of the disease greatly extended. The term Borna disease, as used in the past by continental writers, has in all probability covered more than one disease, but the investigations published from 1924 to 1927 by foreign workers show, beyond doubt, that the term should be reserved for an enzootic equine encephalitis caused by a filterable virus. We understand that when these findings were first reported regarding Borna disease, Professor Gaiger followed the same lines of investigation in Scotland for "grass disease," but was unable to demonstrate any filterable virus in "grass disease," though these experiments have not been published. No other worker in this country has as yet been able to demonstrate a filter-passing virus in "grass disease." Consequently, owing to researches carried out since 1921 when Professor Gaiger made the comparison, "grass disease" must now be definitely concluded as distinct from Borna disease.

An interesting feature of the comparison between these two conditions was the frequent presence, in both, of a diplostreptococcus in the cerebro-spinal fluid, sometimes in pure culture, and highly pathogenic upon inoculation into rabbits, but in this country it was never claimed that this organism was the actual cause.

The botulism theory was put forward at the 1922 Annual Meeting of the Scottish Branch in Perth, though reports in the farming papers in previous years had made many familiar with the findings. That meeting was largely attended and the paper and discussion lasted five hours. From that time the botulism theory of "grass disease" steadily waned, and to-day there is little reason to doubt that "grass disease" is not equine botulism. Readers who are interested in comparing these two conditions should carefully study, in next week's issue of the *Record*, Mr. Nairn's contribution to the discussion at the recent meeting. This observer marshals his facts in a manner which excites admiration. Other reasons than those given by Mr. Nairn for his conclusions will be found by our readers in the *Lancet* for November 19th, 1927.

Professor Pool, in his paper, presents a useful review of some of the literature on "grass disease" and allied conditions, but gives no views of his own concerning the disease. This is probably wise, when just commencing further efforts to solve the problem. Professor Russell Greig makes a most praiseworthy effort to throw light on the nature of the symptoms of the disease.

Readers will be struck by the fact that, though a very great deal of the ground covered by the recent meeting of the Scottish Branch had already been covered by the meetings of 1921 and 1922, no mention is made of the Branch ever having heard of "grass disease" at a previous meeting.

On the whole, from all the work that has been done upon "grass disease," Borna disease, botulism, etc., one may now be justified in regarding "grass disease" as an entity. The cause would seem to be toxic, but that is as far as one can go. Many speakers at the recent meeting dwelt upon the *ingestion* of some toxin, but may a toxin not be elaborated in the alimentary canal without being ingested from some outside source?

The lack of a specific diagnostic test makes the diagnosis of the disease, in isolated cases, far removed from "grass disease" areas, open to question. Unless the clinical picture is very complete, few experienced men would venture a diagnosis, in such circumstances, in horses. Much less would they be inclined to say that this disease occurs in cattle.

(*Continued from previous page*).

although the symptoms of botulinus intoxication bear some resemblance to "grass disease," and the pharmacology of the botulinus group of toxins requires investigation.

(*d*) *Parasital Toxins*: No protozoal or other parasital toxin of animal origin is at present known to possess this action.

(*e*) *Sympathico-mimetic Amines*: As the result of liver disfunction certain amino bases, notably tyramine and phenyl-ethyl-amine, may be liberated in the imperfect breakdown of their respective amino-acids. They produce the effect of sympathetic stimulation (sympathico-mimetic), and, although their action is less powerful than that of adrenaline, it seems possible that their continued action may produce the sympathicotonia of "grass disease." If this be so it postulates a specific primary toxin or animate virus which is capable of hindering the normal deaminization of these toxic metabolites in the liver.

A histological study of the liver in acute "grass disease" therefore suggests itself.

In offering this attempt to interpret the symptoms of acute "grass disease," I confess I do so with some reluctance; but our knowledge of the pathogenesis of the disease is still so meagre that the expression of any fresh view usually meets with some measure of consideration. I therefore present these tentative opinions in the hope that, if they do not help in suggesting a clue to the etiology, they may, perhaps, lead to a clearer understanding of the nature of the disease.

(*To be concluded with a report of the discussion on these papers, which will appear next week.*—Ed.)

CLINICAL AND CASE REPORTS.

Retention of Urine and Rupture of the Bladder in Male Bovines.

By F. T. HARVEY, F.R.C.V.S., St. Columb.

The case "Perforation of the Bladder in a Steer," recorded by Mr. Smythe, of Camborne, in the *Record* of December 24th is of some clinical interest.

Mr. Smythe is unable to throw light as to the cause of what he terms a "perforation," but if we regard the case as one of rupture of the bladder the etiology might emerge. From past experiences of these cases one would have expected to have found, on a rectal examination of the steer on November 29th, a hugely distended bladder, and at the ensuing *post-mortem* examination a calculus lodged within the urethra at the point of the commencement of the S-shaped curve. I have noted on two or three occasions the striking resemblance the bladder presents, some days after rupture, to what one would expect to find in an injury produced by some body such as a pencil, or penholder, having passed through its wall from within. The organ at the time of rupture has been stretched to the utmost, while at the *post-mortem* it is shrunken and collapsed. Mr. Smythe's description of the history and clinical features is quite typical of this class of case, of which in the course of practice I have seen a fair number.

One of the most curious features of urinary troubles in animals is the erratic manner in which cases turn up, especially in male ruminants. I have seen no case for five or six years, but some ten years since I had to operate on three cases within a few weeks, all occurring in fattening cattle.

The cases usually begin with colic and suppression of urine, either partial or complete. In the former there will be a continuous drip of urine from the sheath opening. Diagnosis is easy on rectal examination. Every case of colic in a male bovine should be regarded as a potential case of calculus and demands a careful examination of the urinary apparatus. To linger over the diagnosis is fatal, since promptness in affording relief is the keynote in treatment. Under no circumstances can delay be warranted.

Another point in these cases and a more serious one, is the length of time retention may have been present, and that in a marked degree, without the animal having shown any definite clinical signs or symptoms, and rupture of the bladder may have already taken place ere anything is known to be amiss. I have seen several of these cases. The animal when first seen may be passing into a state of collapse and the "facies hippocratica" becoming well established—pinched expression of the face, with sunken eyes and hollow-cheeks, etc.—the abdomen greatly distended, the prepuce or sheath dry and not easily felt. If the fingers of one hand are placed over the distended parts of the abdomen and a similar point on the opposite side be gently percussed, a distinct thrill or impulse is at once felt if fluid is present in the peritoneal cavity. In these cases it is probable that a small calculus has been lodged for some time at the S-bend, and that the bladder has only been partly emptied, its walls having been kept continually on the stretch. In man a similar condition has been termed "retention over-flow" and here again a considerable distension of the bladder may exist with comparatively little pain. I have known a second stone to pass down, and completely block the channel and quickly determine rupture.

Any colic present ceases when rupture occurs, and it is astonishing the amount of urine which may be found in the peritoneal cavity. If one makes an incision on the left flank it is usually quite easy to locate the opening in the fundus of the bladder. I have found it impossible to deal with the opening by suture and think closing by ligature the only possible plan. One would, of course, also have to arrange for draining the peritoneum and, incidentally, perform urethrotomy at the ischial arch or at the point of lodgment of the stone. The outlook, both surgically and economically, is, of course, grave.

OPERATION FOR RELIEF OF RETENTION.

In fattening animals I have ignored the calculus lodged at the bend, and cut down on the urethra behind and immediately below the ischial arch. Separate the urethra carefully from its supporting tissue for a short distance, draw it upwards a little with the finger as a hook, and cut it across. Divide the freed portion of the urethra symmetrically by means of a director, and suture, if possible, the divided ends to the upper margin of the wound close to the skin, taking care that the mucous membrane be properly fixed. Urine may not flow immediately after operation, but relief has always occurred in an hour or two. I have seen excellent results from the operation.

Traumatic Indigestion.

By R. R. MOODY, M.R.C.V.S., Rothesay.

I have carefully read Professor Imrie's paper in the *Record* of December 10th, and I can only say that in my opinion it is a work of art and will be of inestimable value to every country practitioner in the land. Personally, I have been endeavouring to try and diagnose traumatic indigestion before the heart lesions have developed. Last year I had three cases of impaction of the rumen that I can clearly recall. When the animals did not respond to the ordinary treatment for impaction, I naturally suspected a foreign body, but then I had to wait until the symptoms of heart lesions developed. In two of the cases peritoneal tympany was observed, but unfortunately I did not realise its significance. I did not make a *post-mortem* examination in these cases.

The following cases are to me typical of old-time treatment and the new method as advocated by Professor Imrie. In the first case, treatment was long, tedious and unsuccessful. In the second case the treatment was simple and the results gratifying.

CASE (1). Subject, Ayrshire cow, two or three weeks calved.

5th April, 1927.—Owner noticed cow not feeding as usual and off her milk. He called on me for a bottle of linseed oil and for stomach powders, which he administered himself.

9th April.—Requested to see the animal, I found, on examination, the usual symptoms of impaction of the rumen, *viz.*, appetite much impaired, rumination entirely

suppressed, bowels constipated, milk secretion greatly reduced, temperature 104°F., pulse 96, rumen well filled with food of doughy consistence, while a certain amount of gas appeared to be present between the rumen and the abdominal wall. A purge was given and tonics and stimulants were left.

11th April.—Cow much the same. Salt and formalin in four gallons of water were given by the stomach tube.

12th April.—Salt and formalin given per stomach tube. Tonics and stimulants left.

As on previous occasions, a careful examination was made of the heart, etc., for a foreign body, but I failed to find any diagnostic symptoms.

14th April.—The animal already much reduced in condition and having eaten practically nothing for ten days, I decided to perform rumenotomy. Contrary to my expectations, the food which filled the rumen was quite moist and easily removed. A large quantity was taken away, and an exploration made for a foreign body without success.

15th April.—Cow appeared a little brighter, her appetite had improved and she took some gruel drinks.

18th April.—Owner reported swellings under the belly. On visiting the premises I found the animal showing definite symptoms of traumatic pericarditis. Advised immediate slaughter. On opening into the pericardial sac, which contained a large quantity of fluid, I found a piece of wire about four inches in length lying free in the cavity.

Case (2). Subject, Red Poll cow, three months pregnant.

25th November, 1927.—The owner, a very observant person, called on me for some powders for his cow, which he described as showing the following symptoms: animal not feeding as well as usual, groaning or grunting as if in pain, milk reduced in quantity, not cudding, and stiff when moved.

26th November.—Was requested to see cow. Found animal much the same as described above, *viz.*, temperature 103°F., pulse a little above normal, rumen very full, a suppressed grunt and breathing slightly quickened. The animal was made to move, but I failed to find any evidence of stiffness and the owner agreed she was not as stiff as on the previous day. Rumination was entirely suppressed and milk secretion reduced from 20 to 12 pints. I instructed the owner to put the animal " on the boards " as advocated by Professor Imrie. The cow was only " on the boards " a little over an hour when she commenced to cud.

27th November.—Cow much better, cudding normally, appetite improved, grunting only at intervals, temperature 102°F., pulse normal.

The animal was " on the boards " for four days, when the grunt ceased and she appeared quite normal. On the 5th day (30th November) the owner took it upon himself to remove the boards, but in two hours he replaced them as the cow again commenced to grunt.

3rd December.—Owner happened to be in byre when the cow passed some dung and then commenced to strain. After straining a little she passed about half a pint of blood clot and mucous and then stopped straining.

At the tenth day from commencement of illness the boards were removed and the animal made a complete recovery. I only wish now Professor Imrie had published his paper a year ago, as I might have saved some valuable cows.

ABSTRACTS.

[**Studies of Experimental Swine Plague.** M. P. A. Bel, in a Thesis for the Doctorate of Veterinary Science, Lyons, 1926. Ex *Rev. Vét.*, July, 1927.]

Of all the red diseases of the pig, swing plague is the gravest and most contagious. It may decimate pig-breeding establishments and cause immense losses.

Two forms are recognised from the clinical point of view: one hyper-acute, the other acute. The first is characterised especially by congestive and hæmorrhagic lesions on most of the organs. In the second, the most important alterations are situated in the intestine: the ilio-cæcal region shows erosions, ulcers and pimples on the surface of the mucous membrane.

Almost all the parts of the organism are virulent. The virus seems, however, to proliferate especially in the superficial layers of the mucous membrane and in the endothelium of the capillaries. Experiments have succeeded in weakening it, but hitherto it has been found impossible to use the attenuated virus for vaccination.

The researches of Dorset, MacBride and Niles have established that, by hyperimmunising animals cured of the natural or experimental infection, an active serum may be obtained which produces passive immunity. Its duration is infortunately short in practice, since it does not last more than three weeks.

On the other hand, simultaneous inoculation of the virus and the serum gives an active immunity which protects the subject during its whole economic life. But this method requires care since it is difficult to titrate the respective activity of the serum and the virus. This difficulty explains failures. R. L. Higgins.

[**Concerning the Origin and Nature of Osteoblasts.** Keith, Sir A. *Proceedings of the Royal Society of Medicine.* Vol. xxi. No. 2. December, 1927.].

Three specimens are described which illustrate the formation of true bone in laparotomy wounds of the epigastric region of the abdomen. The literature dealing with their occurrence is surveyed and it is found that the majority of instances (34 out of 36) occur in laparotomy wounds made in or near the supra-umbilical part of the linea alba. Specimens in the R.C.S. Museum are enumerated illustrating heterotopic formation of bone in organs and parts which have no direct connexion with the skeletal system. An attempt is made to explain their occurrence on the traditional conception of bone-growth, namely, that it is the work of a specialised cell normally confined to the skeletal system. It is concluded that the heterotopic formation of bone cannot be explained unless it be accepted, as is now advocated by Professor Leriche, of Strassburg, that under certain states cells of tissues other than the skeletal can become osteoblastic in nature and in action—a view supported by the results of many recent experimental investigations. The view advocated here, not a novel one, is that " new " osteoblasts arise from the capillary system—especially from buds thrown out by that system when a neighbouring substance or body has to be absorbed. Certain necessary conditions are mentioned, but it is admitted that a full and satisfactory explanation of why supra-umbilical scars are more frequently the site of

heterotopic bone formation than laparotomy scars in other parts of the abdomen has not been reached.

[Temporary Sterilisation by Insulin. E. VOGT (*Zentralbl. f. Gynak.*, September 17th, 1927, p. 2436).—Ex. *Brit. Med. Journ.*].

Vogt has found that two or three daily injections of insulin spread over four weeks destroyed sexual desire in female rabbits, and microscopical examination of their ovaries showed an abnormal sparseness of ripening and ripened follicles. Riddle, by administration of small doses of insulin, has been able to prevent ovulation in birds. Previous reports have been published on experimental hormonic sterilisation by transplantation of ovaries from pregnant animals, by injection of extracts of such ovaries, and by injection of placental extracts. Dittler, by intravenous injection of spermatic fluid, induced temporary sterility lasting four months in rabbits. Vogt thinks that such experiments may find an important practical application in the human subject. Temporary sterilisation, for example, in cases of tuberculosis, might be more conveniently and safely induced by insulin treatment than by operation or small x-ray applications. That in the reverse fashion the ovarian internal secretion may influence that of the pancreas is suggested by an observation of Kupferberg (ibid., p. 2438), who found that after applications of radium for carcinoma of the uterus a patient became cured of diabetes, and has since treated several cases of diabetes by introduction of radium into the vagina. Diabetes in a male is reported to have been influenced favourably by placing radium within the rectum.

IMPORTANT REPORT ON BRADFORD MILK.

The Veterinary Inspector for Bradford (Mr. W. Halstead) reports that there are 3,322 dairy cows in the city, housed in 258 dairy farms. The amount of milk produced daily is 7,500 gallons, while about 7,000 gallons are brought into Bradford daily by train or road.

In his 674 visits to the city herds Mr. Halstead discovered twenty-one tuberculous cows, all of which were slaughtered. The owners received £125 19s. 6d. compensation, the salvage on the carcases amounting to £70 13s. 1d.

Only one herd is producing Grade A milk, the output being thirty-eight gallons daily.

Great credit is due to the dairymen of Bradford for the general quality of milk they supply. It is stated that of the 311 samples examined for tuberculosis by the City Bacteriologist, taken from 171 sources of supply—141 inside and thirty outside the city—thirty-two positive results were obtained affecting thirteen sources of supply, all of which were from outside the city.

The result is of some moment, as showing that over 9 per cent. of the sources of milk outside the city is infected with tuberculosis.

The more frequent and exacting veterinary inspection of the city herds, it is stated, is the chief cause of the much better result in city-produced milk.

The 257 samples of milk submitted for bacterical counts only were in 142 cases from outside the city sources of supply and in eighteen those inside the city.

In fifty-two cases the examination showed the need of greater cleanliness at the source, and all these cases were from outside the city, again a remarkable result.—*The Dairy*, 17/12/27.

BRANCH REPORT.

Annual Meeting of the Scottish Branch.*

SUCCESSFUL GATHERING AT GLENEAGLES.

The Annual Meeting of the Scottish Branch was held at Gleneagles Hotel, Perthshire, on Saturday, 15th October, 1927, under the presidency of Mr. Neil H. McAlister, of Kirkcudbright.

About sixty of the members attended luncheon in the Golf Club House previous to the commencement of the General Meeting.

The following gentlemen signed the attendance book :—Messrs. D. Brown, T. M. Inglis, H. W. Robson, Duncan Cumming, W. Nairn and S. Taylor ; Professor A. J. Clark, Dr. Malcolm Wilson and Dr. H. Dryerre of Edinburgh University ; Messrs. W. Anderson, H. M. Wilson, J. J. Macgowan, J. Mitchell, A. Brownlee, D. B. McClure, Professor W. M. Mitchell, Professor A. W. Robb, Messrs. S. Sharp, W. Gardner, G. W. Weir, Jas. MacFarlane, J. Gibson, Jas. Anderson, A. Spreull, J. Brown (Perth), J. Brown (Invergordon), W. Brown (Aberdeen), A. Gofton, Professor A. W. Pool, Professor J. Russell Greig, Messrs. H. Begg, J. G. McGregor, Geo. Howie, Jas. McAllan, J. J. Fergusson, J. R. Baillie, P. Wilson, J. B. Russell, G. W. Begg, R. Reid, A. R. C. Bowie, A. McK. Sievwright, H. McVean, R. Mitchell, Wm. Harris, P. McLeish, Thos. Dalling, Wm. S. Gordon, B. Philip, Geo. F. Boddie, G. D. Munro, Esmond Brown, A. Douglas, H. C. Watson, J. McFarlane, R. S. Marshall, A. Sillars, W. R. Ferguson, D. R. Wilson, H. Preston, and W. C. Miller (Hon. Secretary).

There was a considerable number of others present who unfortunately, did not sign the attendance book. Altogether a total of eighty-three members and others were present at the meeting.

Minutes.—The minutes of the last meeting, having been printed in the *Veterinary Record*, were taken as read.

Apologies.—Apologies for absence were intimated from Dr. O. Charnock Bradley, Professor S. Gaiger, Dr. Buchannan, Mr. W. Wallace Peggie, Professor Whitehouse, Colonel E. J. Butler, Mr. A. Linton Robertson, Colonel McKendrick, Dr. Andrews, Dr. W. G. Smith, Messrs. E. J. H. Sewell and J. N. Ritchie, Professor R. G. Linton, Mr. W. T. Hewetson and Dr. White.

Mr. J. Anderson intimated that the President of the N.V.M.A. wished him to convey an apology to the members present for his unavoidable absence, and to extend his best wishes for a successful meeting.

The PRESIDENT moved that a sincere vote of sympathy be extended to Mrs. Howie and family on the loss they had recently sustained through the death of Mr. Geo. Howie, who for so many years had acted as Secretary of the Scottish Branch. He felt sure that the many members who knew Mr. Howie would agree that it was fitting that some permanent record should be included in the minutes of the Branch, indicating the high esteem in which Mr. Howie was held by all, and the sincere regrets his death had caused.

Mr. WILLIAM BROWN, of Aberdeen, seconded the motion, and paid a high tribute to the regard in which Mr. Howie was held by the practising veterinary surgeons in the North.

* Received on 27th December, 1927.

The motion was duly carried and in respect for the late Mr. Howie one minute's silence was observed, the members meantime standing.

ELECTION OF OFFICE-BEARERS.

The PRESIDENT said that in accordance with custom it was his duty to nominate the President of the Scottish Branch for the forthcoming year. He had much pleasure in putting forward the name of the President of the Ayrshire Division—Mr. W. Gardner, of Maybole.

Mr. J. BROWN seconded, and Mr. Gardner was declared duly elected.

For the office of Secretary, the PRESIDENT proposed Mr. Wm. C. Miller and Mr. A. SPREULL seconded. After election, Mr. MILLER briefly thanked the members.

The PRESIDENT, in a short address, returned thanks to the members of the Scottish Branch for the honour they conferred upon him last year in his absence, when they elected him President for the session. He touched upon the usefulness of Branch Meetings as a means of calling together Scottish practitioners, and said that he was extremely gratified to see present veterinary surgeons from as far north as Invergordon, and as far south as the English Borders. He sincerely wished the Scottish Branch every success, and hoped that it would continue to be attended as well or better than it was that day.

In few words the President then introduced Professor A. W. Pool, the Director of the Animal Diseases Research Association, and Professor J. Russell Greig, of the Royal (Dick) Veterinary College, and called upon them to speak to their papers, copies of which had been distributed to the members prior to the meeting.

[These papers (Professor Pool's on "Grass Disease in Horses" and Professor Greig's on "Acute 'Grass Disease': An Interpretation of the Clinical Symptoms," appear at the commencement of this issue. The discussion which ensued will be published next week.—Ed.]

Mr. J. MCALLAN, of Aberdeen, proposed in a few words a very hearty vote of thanks to the contributors of the papers and to the opener of the discussion, Mr. Nairn. This was acceded to in an extremely hearty manner, the CHAIRMAN remarking that seldom had he been at a meeting of the Scottish Branch at which there had been such a good attendance, and never had he been at one when there had been such a good discussion.

Professor POOL briefly replied.

Thereafter, the meeting resolved itself into Committee, the visitors and guests leaving, to consider official business.

A letter was read from the N.V.M.A., regarding the necessity for the formation of a Veterinary Inspectors' Section in connection with each of the Divisions. Since this letter was circulated to each of the Divisions of which the Scottish Branch is formed, it was decided to take no action.

It was decided, on the motion of Mr. G. Weir, that the next meeting should be held at Perth, and, there being no other proposal, Perth was decided upon for 1928. It was further agreed that the meeting should be held in October, as near to the middle of the month as possible, and the subject for discussion was left to be arranged by the President and Secretary.

The proceedings terminated with a vote of thanks to Mr. McAlister for his very able guidance of the meeting as Chairman.

WM. C. MILLER, *Hon. Secretary.*

NOTES AND NEWS.

The Editor will be glad to receive items of professional interest for inclusion in these columns.

Diary of Events.

Jan. 16th—Meeting of the Editorial Committee, N.V.M.A., 4-30 p.m.

Jan. 20th—Annual Meeting of the Mid-West and South Wales Division, at Bristol.

Jan. 25th—Meeting of the Scottish Metropolitan Division, at Edinburgh.

R.S.P.C.A. *versus* MINISTRY OF AGRICULTURE.

In view of the great degree of interest and importance attaching to the case, we shall reproduce in our next issue a full report of the proceedings at Swindon Police Court on January 3rd, when a prosecution was brought by the Royal Society for the Prevention of Cruelty to Animals against two inspectors of the Ministry of Agriculture.

The prosecution alleged cruelty by terrifying, arising out of the slaughter of about 100 cattle following a local outbreak of foot-and-mouth disease. The Bench dismissed the charge, stopping the proceedings after hearing only three out of a score of witnesses for the defence.

R.C.V.S. COUNCIL ELECTION, 1928.

The Voting Papers for the next Council Election (June 7th, 1928) will be issued to overseas members during the present month, and nominations for election should be in the hands of the Secretary as soon as possible. The following members retire by rotation, but are eligible for re-election:—

Professor J. B. Buxton, Mr. J. Clarkson, Mr. G. H. Locke, Sir John M'Fadyean, Mr. W. D. Rees, Dr. Share-Jones, Mr. J. Willett, Professor G. H. Wooldridge.

We understand that the following new nominations have been already sent in:—

Major D. S. Rabagliati, O.B.E., B.Sc., F.R.C.V.S., Dr. A. Leslie Sheather, D.Sc., M.R.C.V.S., Colonel G. K. Walker, C.I.E., O.B.E., V.D., F.R.C.V.S.

A *Record* APPRECIATION.

The following is typical of a number of seasonable messages of goodwill, embodying appreciation, which have reached us, and for which we tender our most sincere thanks:—"As regards the *Record*, I always await its arrival with keen pleasure, and feel full of gratitude to members of the Editorial Committee and staff for the excellent little paper it now is. There was a time when copies considered useful for future reference were stored away in a little drawer; now, a big drawer is required, as all issues contain much that make it essential that they shall not be mislaid."

R.C.V.S. OBITUARY.

TAYLOR, William Augustus, York Villas, George Street, Broughton, Salford. Graduated, London, 20th April, 1870. Fellow, 7th July, 1877. Died, 6th January, 1928, aged 77 years.

Mr. Taylor's death is recorded with the deepest regret, for the profession in general, and this Association in particular, are deprived by this loss of one who throughout his long and honourable life displayed the greatest interest in the well-being of his profession and took a prominent part in the activities of its organisations. Mr. Taylor, who was head of Peter Taylor and Sons, veterinary surgeons, of Stanley Street, Cheetham, had been President of the Lancashire Veterinary Medical Association, of the Northern Branch of the National Veterinary Medical Association, of the National Medical and Defence Association, and of the National Master Farriers' Association. An expert judge of light horses and hunters, Mr. Taylor was one of the best-known veterinary surgeons in Manchester, where he had practised for over 50 years, while he was a familiar figure at agricultural shows.

Mr. Taylor leaves eight children, but there is no member of the family to carry on his profession, and his death brings to an end a family association with veterinary surgery of over 100 years.

Personal.

Will.—Mr. John Boyd Moore. M.R.C.V.S., of Kilbroyde, Shantallow, Co. Londonderry, retired veterinary surgeon, who died on October 7th, left personal estate in Great Britain and Northern Ireland valued at £9,583. He left:—£5 to the Society for the Prevention of Cruelty to Animals, Londonderry; £20 to his housekeeper, Jane Hunter Laird, if in his service; £50 to his former housekeeper, Isabella Boyd; £50 for the upkeep of the family burying ground.

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Birth. Manley. At Mbala, Uganda, on 20th November, 1927, to Margaret, wife of F. H. Manley, B.V.Sc., M.R.C.V.S., a daughter.

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Appointment. Among the appointments made by the Secretary of State for the Colonies during December was that of Mr. T. B. McClure, M.R.C.V.S., as Veterinary Officer, Kenya Colony.

Road Surfaces and Horse Traffic.

A National Investigation.

At a meeting of a committee of the National Horse Association of Great Britain, appointed to investigate the slippery condition of the roads as affecting horses and cattle, Mr. Cecil G. Argles (Chairman) stated that not only were the main roads dangerous to traffic, but the by-roads were now becoming equally bad. Information had, however, been received as regards a road situated between Alwalton cross roads and Woodston (a suburb of Peterborough) which apparently had proved non-slippery for all forms of traffic. This road had been down at least five years, and there was no record of any accident due to slipperiness.

The committee decided that all the roads which had been reported as non-slippery should be inspected and the evidence carefully scrutinised, while it was decided to invite gentlemen in all counties to send in details of any roads—in use for a considerable time—which had proved non-slippery to horses and cattle. The committee propose to investigate these roads, and to report their findings and recommendations for improvement in the construction and maintenance of roads to a general conference of all horse breeding and other societies representative of all users of horse transport.

It was decided to invite all Masters of Foxhounds to allow a "cap" of one shilling per head to be taken at one meet of their hounds in the near future to defray the expense of the investigation.—*The Times.*

CORRESPONDENCE.

Letters to the Editor should reach the Office not later than by the first post on Tuesday morning for insertion in following Saturday's issue.

All correspondence must bear the name and address of the contributor for publication.

The Editor does not hold himself responsible for the opinions of his correspondents.

Unqualified Assistants.

To the Editor of The Veterinary Record.

Sir,—In each of the last few editions of the *Veterinary Record* there have been one or more letters written appertaining to the profession, under the heading, "What is Wrong, etc." :—

I think myself that the trouble is within the profession, and I fear this letter will bring down upon my head the wrath of many hard-working practitioners.

Bye-law 53 reads: "Any of the following practices on the part of a Veterinary Surgeon is considered by the Council to amount to conduct disgraceful in a professional respect within the meaning of Section 6 of the Veterinary Surgeons Act, 1881. (Here follow Parts I., II. and III.) (IV.).—Meeting an unqualified person in consultation. How many in this profession have unqualified assistants (other than a student) and not only consult with them, but send them out on cases?—thereby contravening also Part III. of the above bye-law, which reads: "The permission by a Veterinary Surgeon for his name to be used by an unqualified or unregistered person or the doing or permitting any other act whereby an unqualified or unregistered person may pass himself off as or practise as a Veterinary Surgeon."

We elect members to Council to protect us and see that these bye-laws are adhered to. Is the blame on the Council?

I should like to hear the opinion of other members of the profession, particularly members of Council, on this question.—Yours faithfully, Garth K. Fenn-Smith.

30 Bryanstone Road,
Bournemouth.
January 4th, 1928.

Tuberculosis Eradication.

To the Editor of The Veterinary Record.

Sir,—The valuable articles which have appeared in the *Record* on this disease or its incidence in Guernsey and Jersey, from the pens respectively of Lieut.-Col. Glasse and Lieut.-Col. Marett, and the excellent paper on Tuberculosis in Children, by Dr. A. Stanley Griffith, should encourage British veterinarians to redouble their efforts to purge Great Britain of it, where it seems more rampant than anywhere else. It would be a much easier task to free this country than it would the U.S.A. with its 68 million head of cattle, besides its numerous other animals subject to the disease.

The permissibility of this plague in our midst is a disgrace to our public health. I have no doubt that if the medical profession spoke and with no divided voice legislation would be introduced to exterminate it ruthlessly and with no qualms of conscience. Medical officers, in general, consider that pasteurised or boiled milk is not a suitable food for infants or invalids and that there should be only one grade of milk: clean milk from non-infected cows. The eradication of clinically tuberculous animals is moonshine and only playing with the malady.—Yours, etc., Henry Gray.

The Editor acknowledges the receipt of the following:—

Report of a meeting of the Dumfries and Galloway Division, from Mr. A. Young (*Hon. Secretary*).

Report of a meeting of the Southern Counties' Division, from M. J. Facer (*Hon. Secretary*).

Communications from Mr. K. Sitaram (Pahang), and Lieut.-Col. T. D. Young (London).

Annual Report of the Civil Veterinary Department, United Provinces, for the year ending 31st March, 1927.

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